Pattern of Five-Year Weight Change by Age and Birth Cohorts in a Swedish Adult Population

Faisal Al-Emrani[1,*,§], Martin Stafström[1,§], Jonas Björk[2,§] and Per-Olof Östergren[1,§]

[1]*Lund University, Social Medicine and Global Health, Department of Clinical Sciences Malmö, Clinical Research Centre, S-205 02 Malmö, Sweden;* [2]*Lund University, Division of Occupational and Environmental Medicine*

Abstract: The driving factors behind the global epidemic of obesity have not yet been fully elucidated. Investigating the effect of age and cohort on weight change might enable us to develop effective intervention strategies.

Aims To analyse the pattern of BMI change by age and birth cohort over five-year follow-up in adult Swedish sample.

Methods The data was drawn from the Scania Public Health Cohort. The baseline survey was conducted between 1999 and 2000, and the follow-up in 2005. A total of 10,373 individuals responded to both surveys; all were included in the present study. The data was stratified into three-year intervals according to a respondent's birth year and age at baseline. This was done in order to allow for age and cohort effect analysis by means of fixed effects linear mixed models.

Results We observed a significant increase in BMI between baseline and follow-up for males and females ($p < 0.001$), as well as significant age and cohort effects resulted from age−period and cohort−period models, respectively, for both genders. The age effect on BMI varied between −1.94 to +1.10 BMI units among males and −3.15 to +0.54 among females. The cohort effect on BMI varied from −1.67 to +1.61 BMI units in males, and from −2.28 to +1.18 in females.

Conclusions Our analyses suggest that cohort effects play a contributory role in obesity. Our results also show that it is difficult to distinguish between age-period and cohort-period effects in our study, due to on the one hand the linear relationship between age, period and cohort, and on the other hand to a short follow-up.

INTRODUCTION

Overweight and obesity are on the rise globally; the Scandinavian countries are no exception [1, 2]. High body mass index (BMI) has been shown to be a major risk factor for an array of chronic illnesses, such as hypertension, diabetes, musculoskeletal disorders, and some types of cancer [3]. In 2004, overweight and obesity contributed 5% to the global burden of disease in terms of Disability Adjusted Life Years (DALY) [4].

The current epidemic of overweight and obesity has a great number of determinants, e.g., diet [5], lifestyle [6], technical innovations [7], socioeconomic status [8, 9] level of education [10, 11], physical inactivity [12, 13], and gender [14]. It has been suggested that epigenetic factors also have an impact on BMI. The latter claim is based on the hypothesis that inadequate nutrition during pregnancy switches on genes in the offspring that determine the rate of fat accumulation, even in periods when access to food is unrestricted. It has been postulated that this effect might persist for over a generation [15, 16]. Such a hypothesis could explain why immigrants especially those from low-income countries have a greater risk of developing obesity than the high-income indigenous population of their adoptive home [17].

The mechanisms cited above might affect weight gain over the life cycle of an individual, or could be manifestations of changes in the environment during a specific time period, e.g., when economic policies modify the cost and availability of food. Additional factors may only affect certain cohorts of individuals, depending on eating habits, food preferences, or the epigenetic possibility mentioned. These effects require different intervention strategies. Therefore, it would be useful to better understand the role of each element in the overweight epidemic. Combinations of factors could also be expected to vary considerably by geographical location and between different sub-groups of a given population over time.

The temporal effects on trends of weight change are of great relevance in a health equity perspective. Previous researchers have acknowledged this situation and focused on the following elements: (a) age effects (variation due to physical and social life changes), (b) period effects (variation over time due to environmental factors), and (c) birth cohort effects (variations attributable to exposures during pregnancy or early childhood that are sustained over the course of a life span). The three factors are collectively known as APC effects [18].

*Address correspondence to this author at the Lund University, Social Medicine and Global Health, Department of Clinical Sciences Malmö, Clinical Research Centre, S-205 02 Malmö, Sweden; Tel: (+46)40 391408; Fax: (+46)40 391333; Email: Faisal.al-emrani@med.lu.se

§*These authors contributed equally to this work*

Attempts have been made since the mid-1970s to model these effects for various health problems (e.g., smoking and cancer), both in the way they are differentiated and how they interact [19-21]. In a repeated cross-sectional study among Australian adults, birth cohort affiliation was significantly associated with the prevalence of overweight. The effect was more obvious among those born since 1960 than for those born earlier [22]. Another repeated cross-sectional study in the US indicated that one's birth cohort significantly influenced the odds ratio of obesity, since BMI increased in every five-year period between 1976 and 2001 [23].

Several longitudinal studies from the US, Denmark, Korea, and The Netherlands detected period and cohort effects on BMI change and obesity prevalence in all study contexts. Olsen *et al.* found that the obesity epidemic in Denmark has grown in phases [24]. An increase was identified for birth cohorts from the early 1940s to the mid-1950s, and from the late 1960s onward. A recent Korean investigation concluded that there are period and cohort effects for diabetes as well as obesity. The effect, however, was less pronounced in the case of diabetes [25]. Continuing research on the overweight and obesity epidemic suggests that data on period and birth cohort effects have had little impact on the search for the cause of this global issue. Intervention strategies appear to be even less affected by such findings, although studies taking period and birth cohort effects into account have shown their substantial influence.

The present paper seeks to investigate the pattern of BMI change by age and birth cohort over five-year follow-up in adult Swedish sample: the Scania Public Health Cohort (SPHC).

To the best of our knowledge, ours is the first study to apply this strategy to a Swedish population sample. We believe the results will focus the attention of decision makers and health policy planners on less acknowledged causes, thereby allowing them to intervene at the family, society, or national level and better address the equity aspect of the overweight epidemic.

METHODS

Population and Instrument

The data set of this study is derived from two surveys: the 1999 baseline assessment and 2005 follow-up SPHC questionnaire distributed to a representative sample of the general population in Scania, Sweden's southernmost county.

In 1999, 24,922 males and females ages 18 to 80 were randomly selected within a non-proportional geographically stratified sample. The stratification was done by a random selection from each of the 60 administrative units in the county of Scania. A questionnaire was mailed to these individuals, and their responses were collected between November 1999 and April 2000. After three reminders, 13,604 (59%) had responded to the survey. In 2005, the same questionnaire was again mailed to those who had completed it in 1999. This time a total of 10,485 (77%) responded. However, due to incomplete or questionable data (such as missing height or weight, or a large discrepancy in the height reported in the two surveys), 102 people were excluded. The

final cohort was comprised of 10,373 individuals, of whom 4,584 (43.8%) were males and 5,789 (55.3%) females.

The SPHC questionnaire had 12 sub-sections covering a wide range of issues, including self-rated health, health care utilization, environmental and occupational factors, health-related behaviours, and socio-demographic information. A fuller description of SPHC may be found in [43].

Measurements

Height and weight were self-reported and specified in the questionnaire as "height (in cms) without shoes" and "weight (in kgs) without clothing". BMI was calculated by dividing weight in kilograms by height in squared meters (kg/m^2).

Overweight and obesity were defined according to World Health Organization standards as overweight = BMI 25.00 to 29.99 kg/m^2, and obesity = BMI $\geq$ 30 kg/m^2 [3].

Age was established by means of the official Swedish population register.

Statistical Analysis

Analyses were performed using Predictive Analytics Software (PASW) 18.0 for Windows with a 5% level of significance.

Dependent Variables

Birth cohorts were defined by year of birth in three-year intervals. Respondents were subsequently classified into 21 birth cohorts. At the same time, 21 age groups were formed, by stratifying the respondent's age into three year interval. The age and cohort effects on BMI change between baseline and follow-up were estimated using linear mixed models (LMM). The cohort-period (CP) and age-period (AP) models have been fitted to the data analysis with the baseline period and oldest birth cohort, and oldest age group, respectively, as reference. BMI, as a repeated measure, was analysed as the dependent variable, while birth cohorts or age groups, and year of analysis (1999/2000 and 2005) were added as fixed factors and fitted into the LMM with a 95% confidence interval. The same strategy was then used to evaluate the AP effects**.** We also analyzed the interaction between birth cohort and period & between age and period using the same models.

Ethical Statement

This research has been carried out in accordance to the Helsinki Declaration of research ethics regarding human subjects. Both assessments of the cohort have been approved by the regional Research Ethics Committee in Lund University (D nr 1999-99 and 2005-471).

RESULTS

The proportion of overweight and obese individuals in the study population increased between the baseline and follow-up surveys. The prevalence of overweight rose among males from 43.3% to 45.1%, and among females from 27.5% to 29.5%. At the same time, the prevalence of obesity went from 10.2% to 12.5% among males and from 9.6% to 12.6%

among females. The increase in overweight was significant for both sexes, while the change in obesity was only significant for females (*p* = 0.037) (Table **1**).

We also analyzed the change of mean BMI by gender and found that mean weight had increased significantly over the five years surveyed. Mean BMI increased from 25.7 to 26.1 among males and from 24.5 to 25.0 among females. This increase in BMI was generally significant when we stratified the sample based on three-year birth cohorts, with the exception of the oldest birth cohorts, where decreases or no changes in BMI were noted (Table **2**).

The cohort effects, resulted from cohort period models, i.e., the variability in BMI across birth cohorts adjusted for the period effect, ranged from –1.19 BMI units among males born from 1979 to 1981, to +1.84 for males born from 1937 to 1939. In females the cohort effects ranged from –1.63 among women born from 1979 to 1981, to +1.48 among women born from 1934 to 1936. The age effects resulted from age period model show, overall, similar variability. (See Table **3**).

Results from interaction models suggested that the change in BMI between baseline and follow-up differs depending on birth cohort among both males and females in the cohort-period model (p<0.001 for both interactions). In the age-period model the change in BMI differs depending on age among females (p=0.001 for interaction), but apparently not significantly among males (p=0.28).

In (Figs. **1** and **2**) we plotted the cohort- and age-specific changes in BMI between baseline and follow up for each three-year-interval birth cohort and age group (BMI change and 95% CI) stratified by gender. The cohort-specific changes in BMI seemed to follow a linear trend among both males and females, with the clear increase in BMI observed among the youngest birth cohorts gradually declining and eventually becoming negative among the older cohorts. The age-specific changes in BMI was positive for most age groups among both males and females, suggesting that study participants of the same age generally had higher BMI at the follow up than at baseline, but with no consistent trend across the age groups.

DISCUSSION

Our findings show that mean BMI increased in the population studied between 1999/2000 and 2005, which could be due to period, age or cohort effect.

Results from cohort-period and age-period models have shown BMI change in both males and females in nearly all the cohorts and age groups studied; increase in younger and middle ages and decline in older segments of the population. Some birth cohorts and age groups showed a greater increase in BMI than others, both in males and females. For example, the male cohorts born in 1928 to 1933 showed a smaller weight gain compared with the cohorts born immediately before and after. The explanation for these discrepancies in the BMI scores across birth cohorts between baseline and follow-up might be related to events that have happened in early life.

Overall, the age effects show a similar pattern with the cohort effects, that make it difficult to distinguish between age-period and cohort-period effects in our study, due to on the one hand the linear relationship between age, period and cohort, and on the other hand to short follow-up.

The interaction analyses in both models (cohort-period and age-period) have shown a significant BMI change depending on birth cohort for both genders while with age only among females, which indicated that the effect of period is more pronounce among females than males.

As with many other countries in the Organisation for Economic Cooperation and Development (OECD), Sweden has seen a rapid increase of overweight and obesity in the general population over the past three decades. It is estimated that the prevalence of obesity in adults has doubled within the last twenty years, and is now approximately 10% in males and females [26, 27]. Our findings suggest that these figures have already been exceeded.

The results presented here are similar to those found in Denmark [28], Norway [29], Finland [30], France [31], Australia [22, 32], and the US [32]. There is agreement that weight increases throughout the life course until about age 60. A stable pattern in birth cohorts between baseline and follow-up has also been demonstrated by these studies. However, our findings suggest that female birth cohorts followed a different pattern that has not been substantiated by other research.

We began at baseline with a relatively large proportion of subjects above age 65 and noted how their BMI score between baseline and follow-up tended to decrease. We assume that this was mainly the result of physiological

Table 1. Descriptive Data of the Sample: (*n* and %), Prevalence of Overweight, and Obesity (Stratified by Gender)

			1999		2005		P value
			n	%	n	%	
Males			4584	43.8	4369	44.1	0.984
Females			5789	55.3	5528	55.9	
Overweight		Males	1985	43.3	2060	45.1	0.000
		Females	1590	27.5	1703	29.5	0.000
Obesity		Males	466	10.2	575	12.5	0.581
		Females	558	09.6	728	12.6	0.037

Table 2.   Crude Difference in BMI Between Baseline and Follow-Up; Mean, Standard Deviation, BMI Difference, and t-test, Categorized by Gender and Three-Year Birth Cohort Interval

Birth Cohorts	Males						Females					
	Mean BMI 99	SD	Mean BM 05	SD	BMI change	*p* value	Mean BMI 99	SD	Mean BMI 05	SD	BMI change	*p* value
1979–1981	22.68	2.96	24.00	3.14	1.32	0.00	22.03	3.15	23.38	4.24	1.35	0.00
1976–1978	23.98	3.80	25.38	4.19	1.40	0.00	22.56	3.50	23.50	4.09	0.94	0.00
1973–1975	24.22	2.93	25.31	3.38	1.09	0.00	23.80	4.70	24.94	5.21	1.14	0.00
1970–1972	25.36	3.81	26.33	4.08	0.97	0.00	23.44	4.05	24.29	4.47	0.85	0.00
1967–1969	25.69	3.81	26.49	4.12	0.80	0.03	23.70	4.02	24.47	4.46	0.78	0.30
1964–1966	25.59	3.44	26.35	3.86	0.75	0.38	24.04	4.09	24.96	4.71	0.91	0.18
1961–1963	25.80	3.18	26.37	3.64	0.57	0.00	24.05	3.82	24.82	4.38	0.77	0.00
1958–1960	25.78	3.43	26.11	3.73	0.33	0.01	24.27	4.24	24.78	4.15	0.51	0.00
1955–1957	25.64	3.20	26.28	3.43	0.64	0.00	24.04	3.66	24.96	4.01	0.92	0.00
1952–1954	25.79	3.32	26.25	3.48	0.46	0.00	24.40	3.80	25.06	4.21	0.66	0.00
1949–1951	25.99	3.56	26.34	3.48	0.35	0.00	24.56	4.14	24.99	4.45	0.42	0.00
1946–1948	26.10	3.30	26.40	3.59	0.30	0.00	25.18	4.09	25.60	4.50	0.42	0.00
1943–1945	26.04	3.58	26.30	3.66	0.25	0.02	24.78	4.26	25.13	4.43	0.35	0.00
1940–1942	26.29	3.00	26.46	3.20	0.17	0.10	24.97	3.54	25.42	3.99	0.45	0.00
1937–1939	26.55	3.28	26.75	3.65	0.21	0.07	25.49	3.48	25.79	4.07	0.30	0.01
1934–1936	26.32	3.70	26.35	4.13	0.03	0.82	25.99	3.99	26.11	4.20	0.13	0.31
1931–1933	25.89	3.25	26.13	3.59	0.24	0.05	25.71	3.91	25.76	4.15	0.05	0.71
1928–1930	25.96	3.17	25.77	3.44	-0.19	0.13	25.77	3.90	25.61	3.92	-0.16	0.23
1925–1927	25.95	3.17	25.39	3.31	-0.56	0.00	25.35	3.65	24.94	3.90	-0.41	0.00
1922–1924	25.78	3.27	25.67	3.22	-0.11	0.48	25.31	3.74	24.57	3.82	-0.74	0.00
1919–1921	25.20	2.97	24.52	3.03	-0.69	0.00	24.93	3.66	24.07	4.21	-0.87	0.00

aging, as the birth cohort effects were positive. If weight gain were also found among older age groups, it would most likely lead to a heavier burden on the health system, e.g., increased orthopaedic and diabetes health care needs. The decline of BMI in older cohorts could be related to behavioural and physiological changes within individuals, such as a decrease in energy requirements at rest with advancing years [33]. An increase in weight among younger cohorts and a decrease among older ones is a common finding [34-36]. However, a Dutch longitudinal cohort study reported weight gain among individuals over 60 years of age as well [37].

Several studies have attempted to explain the underlying mechanisms for the discrepancy in weight gain observed among different birth cohort groups. Recent publications on obesity have suggested several factors: changes in the global food system, low caloric energy expenditure due to modern technology, and individual factors, including genetic effects

that result from interaction with the local environment. These might account for the variability in body size between individuals [38, 39]. However, why one gains more BMI units at young age is not clear.

Previous investigations have found it likely to be related to low physical activity. For example, a recent Australian study found that younger birth cohorts of males tended to engage in less leisure time physical activity than older generations [22].

Fast food consumption on a regular basis may be another factor in obesity [40]. Industrial development may be responsible for an increase in energy intake, a diet of low-quality carbohydrates and fats, and a decline in physical activity. This implies that a general change in living conditions plays an important role in the present epidemic of obesity [41].

Table 3.   Cohort and Age Effects Resulted from (Cohort-Period and Age-Period Models) of Linear Mixed Analyses, Categorized by 3-Year Birth Cohort and Age Groups for Males and Females

Birth Cohort	Male				Female			
	Estimate	*p* value	95% CI		Estimate	*p* value	95% CI	
			Lower	Upper			Lower	Upper
Intercept	24.66	<.001	24.00	25.31	24.41	<.001	23.71	25.12
1979–1981	-1.19	.005	-2.02	-0.36	-1.63	<.001	-2.50	-0.77
1976–1978	-0.17	.689	-1.01	0.67	-1.55	.001	-2.43	-0.67
1973–1975	-0.21	.621	-1.06	0.63	-0.22	.612	-1.09	0.64
1970–1972	0.94	.024	0.13	1.75	-0.75	.083	-1.61	0.10
1967–1969	1.28	.002	0.48	2.08	-0.43	.311	-1.27	0.40
1964–1966	1.18	.003	0.41	1.95	-0.17	.688	-0.98	0.65
1961–1963	1.23	.002	0.45	2.01	-0.26	.544	-1.09	0.57
1958–1960	1.15	.004	0.37	1.93	-0.12	.766	-0.94	0.69
1955–1957	1.39	<0.001	0.61	2.18	-0.05	.901	-0.88	0.78
1952–1954	1.26	.001	0.50	2.03	0.11	.786	-0.70	0.93
1949–1951	1.36	<0.001	0.61	2.10	0.14	.731	-0.66	0.94
1946–1948	1.37	<0.001	0.63	2.12	0.68	.096	-0.12	1.48
1943–1945	1.38	<0.001	0.63	2.12	0.32	.429	-0.47	1.12
1940–1942	1.45	<0.001	0.69	2.20	0.65	.127	-0.19	1.49
1937–1939	1.84	<0.001	1.07	2.60	0.94	.026	0.11	1.78
1934–1936	1.38	.001	0.60	2.16	1.48	.001	0.63	2.32
1931–1933	1.05	.010	0.25	1.84	1.06	.014	0.22	1.91
1928–1930	0.67	.098	-0.12	1.46	0.91	.037	0.05	1.77
1925–1927	1.12	.007	0.30	1.94	0.64	.164	-0.26	1.54
1922–1924	0.83	.060	-0.03	1.69	0.64	.165	-0.26	1.54
1919–1921	*Reference				*Reference			
Period effect**	0.38	<0.001	0.33	0.44	0.50	<0.001	0.44	0.55
Age Groups /Intercept	25.15	<0.001	24.81	25.49	24.67	<0.001	24.30	25.15
18–20	-1.92	<0.001	-2.46	-1.37	-2.55	<0.001	-3.12	-1.92
21–23	-1.20	<0.001	-1.73	-0.66	-2.04	<0.001	-2.61	-1.20
24–26	-0.88	<0.001	-1.36	-0.41	-1.42	<0.001	-1.91	-0.88
27–29	-0.10	.671	-0.56	0.36	-1.37	<0.001	-1.86	-0.10
30–32	0.06	.800	-0.38	0.50	-0.73	.002	-1.19	0.06
33–35	0.52	.017	0.09	0.94	-0.78	.001	-1.24	0.52
36–38	0.60	.004	0.19	1.02	-0.50	.028	-0.94	0.60
39–41	0.85	<0.001	0.45	1.26	-0.15	.490	-0.59	0.85

(Table 3) contd….

Age Groups /Intercept	Male				Female			
	Estimate	p value	95% CI		Estimate	p value	95% CI	
			Lower	Upper			Lower	Upper
42–44	0.85	<0.001	0.45	1.25	-0.19	.386	-0.62	0.85
45–47	0.92	<0.001	0.52	1.32	0.03	.875	-0.39	0.92
48–50	1.05	<0.001	0.67	1.43	0.28	.184	-0.13	1.05
51–53	0.96	<0.001	0.57	1.35	0.44	.043	0.01	0.96
54–56	1.10	<0.001	0.74	1.47	0.39	.053	-0.01	1.10
57–59	0.99	<0.001	0.60	1.37	0.71	.001	0.28	0.99
60–62	1.06	<0.001	0.72	1.41	0.54	.005	0.16	1.06
63–65	0.93	<0.001	0.54	1.31	0.95	<0.001	0.52	0.93
66–68	0.92	<0.001	0.60	1.24	0.74	<0.001	0.39	0.92
69–71	0.69	.001	0.29	1.09	0.90	<0.001	0.46	0.69
72–74	0.84	<0.001	0.58	1.10	0.62	<0.001	0.33	0.84
75–77	0.23	.289	-0.20	0.66	0.55	.022	0.08	0.23
78–80	*Reference	0			*Reference	0		
Period effect**	0.29	<0.001	0.23	0.36	0.28	<0.001	0.21	0.29

*Reference group in the models   Period effect**: baseline 1999/2000 as a reference group

Cohort (left) and age (right) specific BMI change in males
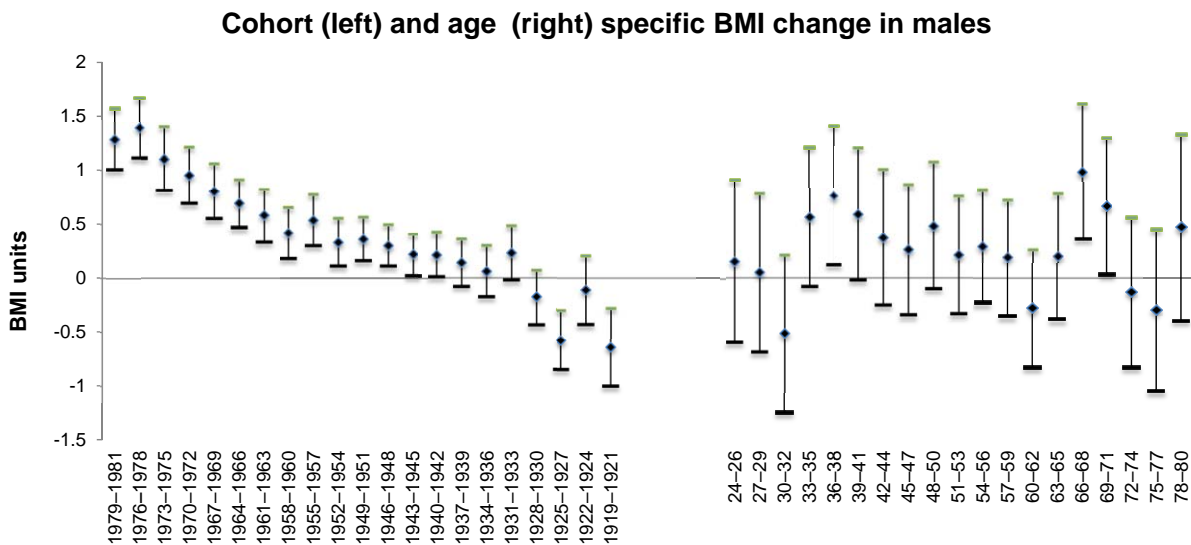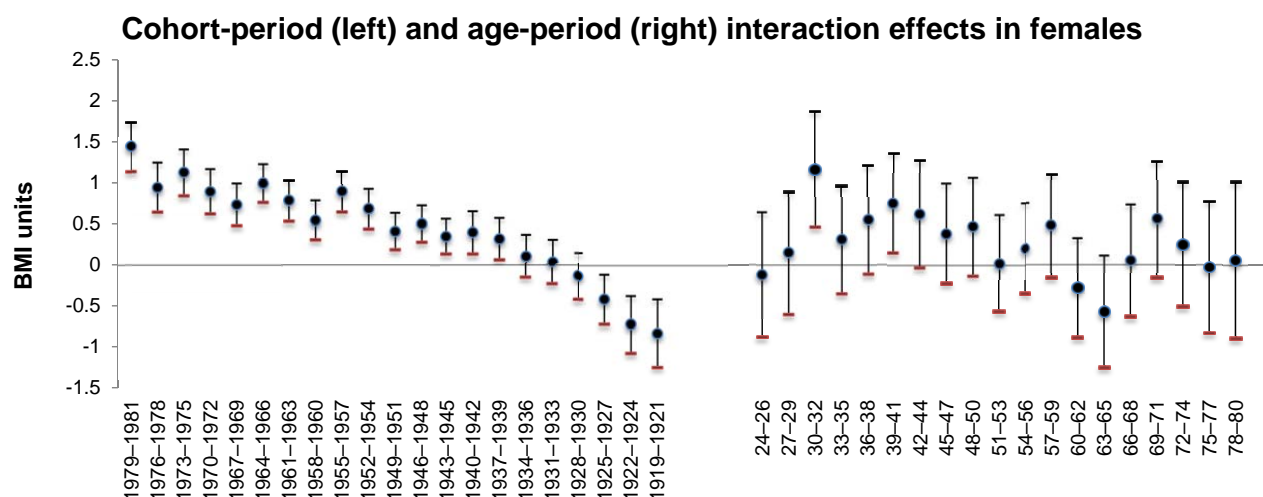

Fig. (1). Cohort- and age-specific changes in BMI from baseline (1999/2000) to follow-up (2005) (BMI units and 95% CI) in males

In our Swedish study, the lack of a diverging pattern regarding cohorts born during WWII may be compared with findings in Dutch and Danish populations [42, 43].

The epigenetic theory would postulate an increase in BMI change for those born during WWII. Since this could not be verified in our study, we therefore conclude that our findings do not support the hypothesis of a epigenitically

induced cohort effect in the general population in southern Sweden.

We believe the present study to be the first that analysis the population of a country not traumatized by direct warfare during this period, and whose population did not experience starvation or near-starvation.

Cohort-period (left) and age-period (right) interaction effects in females

Fig. (2). Cohort- and age-specific changes in BMI from baseline (1999/2000) to follow-up (2005) (BMI units and 95% CI) in females.

STRENGTHS AND LIMITATIONS

Our study has several advantages. First, it is longitudinal, prospective, and population-based, i.e., it examines changes in specific individuals. Second, it analyses a general population across all adult age groups, with baseline measurements ranging from ages 18 to 80. This gave us the opportunity to analyse and compare the pattern of BMI change among a wide range of birth cohorts, whereas most previous studies have focused only on a specific age group or birth cohort, such as adult males or adult females. Third, it includes the study of older individuals ages 65 and above, thus presenting a more complete demographic picture of the overweight and obesity challenges in a high-income country. Fourth, to the best of our knowledge, it is the first attempt to separately evaluate age, period, and cohort effects on BMI trends between baseline and follow-up in a general Swedish population.

At the same time, we are aware of certain limitations. The response rate was 59% for the baseline assessment, which raises the issue of selection bias as an alternative explanation of our findings. However, the discrepancy in socio-demographic characteristics (level of education, income, and country of origin) between respondents and non-respondents has been investigated in a previous study [43], and showed the only difference to be a lower participation rate among individuals born outside Sweden. Moreover, health care utilisation during the year of the survey was very similar in both groups, indicating that a strong selection bias due to general status was improbable. Also, the BMI statistics we used were based on self-reported data, which may have contributed to an underestimation of overweight and obesity prevalence. Finally, another limitation in the method we chose is that it did not allow us to distinguish between age, period, and cohort effect within the same model. Holford (1991) suggested the approach in common use of estimating APC effects by applying a Poisson regression model. Unfortunately, since we only had two data points, this model would not have been appropriate. We have however, been able to disentangle age and cohort

effect by applying age-period and cohort-period models, the same strategy was applied in several longitudinal studies [44, 45].

CONCLUSION

BMI increased during the five-year follow-up in most age groups. Our findings suggest that cohort effects may play a role in obesity, although those effects contradicted the epigenetic hypothesis. Moreover, these results suggest that it is difficult to distinguish between age-period and cohort-period effects in our study, due to on the one hand the linear relationship between age, period and cohort, and on the other hand to short follow-up.

CONFLICT OF INTEREST

The author(s) confirm that this article content has no conflicts of interest.

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AUTHORS' CONTRIBUTIONS

All the authors participated in the design of the study. FA-E and MS performed the statistical analyses in collaboration with JB and FA-E wrote the first draft of the manuscript. P-OÖ was responsible for the data collection. P-OÖ, JB and MS commented on the draft versions of the manuscript. All authors read and approved the final manuscript.

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